

Incidence of Mucooid *Pseudomonas aeruginosa* from Clinical Sources

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The high frequency of pulmonary infection by mucooid strains of *Pseudomonas aeruginosa* in patients with cystic fibrosis can be interpreted to represent an apparent mutation resulting from environmental factors within the host.

Pseudomonas aeruginosa is not usually considered to be an encapsulated microorganism, and it does not normally produce an abundant extracellular polysaccharide (5). Therefore, mucooid strains of this species are rare in nature. However, in a group of 78 patients diagnosed as having

only 0.8% of 242 isolations. Elston and Hoffman (4) isolated 8 mucooid strains or 1.7% of 475 cultures of *P. aeruginosa*. During the past 5 years, 12 mucooid strains or 2.1% of 560 clinical cultures of *P. aeruginosa* have been isolated at the Texas Institute for Rehabilitation and Research,

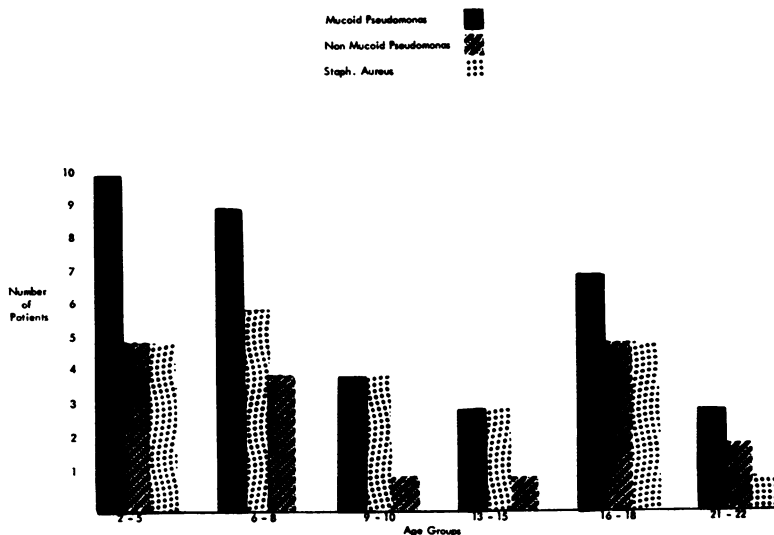


FIG. 1. Contrast between the incidence of pulmonary infection by *P. aeruginosa* and that by *S. aureus* in 36 CF patients expiring primarily as a result of pulmonary insufficiency. This illustrates the predominance of mucooid strains in the pulmonary tree before death. All of the 36 patients harbored *P. aeruginosa* in their sputum.

cystic fibrosis (CF), Doggett et al. found that 70% of their sputum cultures contained strains of *P. aeruginosa* which were mucooid (encapsulated) in colonial morphology (2). In comparing this high incidence of mucooid isolates among CF patients with the findings of other investigators with subjects not having CF but clinically harboring *P. aeruginosa*, the following data were noted. Çetin et al. (1) cultured mucooid strains in

Houston, Tex., from non-CF patients undergoing treatment for various chronic illnesses.

Because of its antibiotic resistance, an increased incidence of *P. aeruginosa* is usually associated with long-term chemotherapy in chronic illnesses. One would, therefore, expect similar frequencies of mucooid strains in chronic disease other than CF if these atypical forms emerge as a result of chemotherapy. As noted above, this is not the

case. The high frequency of pulmonary infection by mucoid strains of *P. aeruginosa* in patients with CF can therefore be interpreted to represent an apparent mutation resulting from environmental factors within the host. It has been shown that the rough form of *P. aeruginosa* precedes the mucoid form in the pulmonary tree (3) and, later, rough, smooth, and mucoid colonies can be simultaneously isolated from the same sputum sample. Eventually the mucoid form predominates in the flora (Fig. 1). Also, pyocin production patterns have shown that mucoid strains do not represent infection by unusual *Pseudomonas* strains but are predominately the commonly encountered strains (B. J. Rosenstein and R. H. Drachman, *Bacteriol. Proc.*, p. 101, 1968).

It is suggested that mucoid strains of *P. aeruginosa* isolated from sputum be morphologically differentiated from the rough or smooth forms. The high incidence of mucoid strains of *P. aeruginosa* in the tracheobronchial tree of patients with CF could possibly aid in detecting this dis-

ease when such microorganisms are cultured, especially when *Staphylococcus aureus* is also present.

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